In addition to chromosomal abnormalities, leukemic cells from AML patients show changes in gene expression. The term *gene expression* can refer to increases or decreases in the biosynthesis of mRNA, and it can also refer to changes in the rate of biosynthesis of the corresponding polypeptides. According to Miller and Stamatoyannopoulos (128), analysis of gene expression using microarrays revealed that poor prognosis in AML patients was associated with the up-regulation of several hundred different genes, such as *BCL11A*, *TBXAS1*, *HOXB5*, and *HOXA10*, while poor prognosis was associated with the down-regulation of several hundred different genes, including *EML4*, *C3AR1*, *SMG1*, and *SEMA3F*. Gene expression analysis can serve as a tool for predicting patient outcome, but it can also identify potential drug targets. The gene expression data were used as a basis for suggesting using *TBXAS1* and *SEMA3F* as drug targets (129).

AML is treated by two therapeutic steps (130). The first step is induction, which results in remission. The second step is postremission therapy, which prevents relapse. Drugs in use for treating AML include clofarabine (purine analog), laromustine (DNA alkylating agent), gemtuzumab ozogamicin (antibody), decitabine (inhibitor of DNA methyltransferase), and azacitidine (inhibitor of DNA methyltransferase) (131).

2. *Acute Promyelocytic Leukemia*

APL is a subset of AML, and comprises about 10% of adults with AML (132). APL manifests itself by spontaneous bleeding (133). The bleeding is potentially fatal, and it is recommended that treatment be started, after an emergency consultation with a hematologist, before the diagnosis is confirmed. Death can result from bleeding in the central nervous system, lungs, or gastrointestinal tract (134).

APL involves a chromosomal defect, where there is a translocation between chromosomes 15 and 17. This translocation generates the fusion gene involving the PML gene and retinoic acid receptor-alpha gene (RAR-alpha). PML stands for "promyelocyte." The resulting fusion gene and the expressed fusion protein are called PML-RAR-alpha. The fusion protein blocks the differentiation of the cells (135).

The disease is highly curable, where treatment involves all-trans-retinoic acid (a form of vitamin A) plus anthracycline. Patients also

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